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BY DUNCAN MATHESON MACKAY, M.D.,

Honorary Aurist to the Hull, East Yorkshire, and Lincolnshire Institution for the Deaf and Dumb; Late Senior Clinical Assistant to the Hospital for Diseases of the Throat, Golden Square, London, W., etc.

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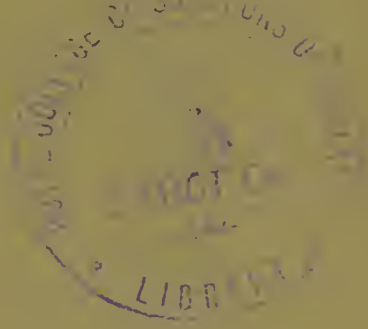

"THE PRACTITIONER," LIMITED,
HOWARD STREET, STRAND, W.C.

THE GENERAL PRACTITIONER AND DEAF MUTISM.[1]

By DUNCAN MATHESON MACKAY, M.D.,

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I HAVE ventured to couple the general practitioner with deaf mutism, because, after all, the problem of deaf mutism is really a problem for the practitioner of general medicine rather than for the practitioner of aural surgery. The victim of deaf mutism may, in the end, come to one who professes a specialty, but it is usually at a time when no help, of the kind he seeks, can be given him. Standing, as he does, between two, the general practitioner and the educationalist, the aurist has too often to confess that he is helpless. Whoever saw the patient earlier might perhaps have done something, now there is no man to help but the schoolmaster.

For, sorrowfully, one must admit, deaf mutism is most frequently incurable. There is only one method, by which it can be treated with any reasonable hope of success, and that is,—as an Irishman might say,—by treating it before it is there to treat. Which saying is but another way of asserting that, while deaf mutism is, as a rule, incurable, it is, not seldom, preventable.

And of all the practitioners of medicine and surgery, the general practitioner is the one who can do most to prevent it. It is his advice that the public acts upon, when it acts upon expert advice at all in things pertaining to health, and it is his aid that the afflicted public seeks first, when it awakens to a sense of its aural disabilities.

Such being the position of affairs, I am taking it upon me to put together a brief statement of the present views as to the causation of deaf mutism, and a summary of the more important findings in its morbid anatomy and pathology. In doing this, I shall draw largely upon the collected evidence

[1] A Paper read before the Hull Medical Society.

and views, comprised in two volumes, of Kerr Love of Glasgow,[1] and Mygind of Copenhagen.[2]

DEFINITION.

By deaf mutism is meant deafness and dumbness co-existing. The exact degree of the deafness varies with different individuals, but, practically, it may be stated to be that degree, which is sufficient to prevent the sufferer from hearing the voice as used in conversation and teaching. Now, as speech is ordinarily learned by a child's hearing the speech of others, and imitating it, the result of deafness of this degree is that, not having any model for speech to imitate, the child does not learn to speak at all.

In deaf mutism, therefore, with few exceptions, the organs of speech are all present and perfect,—they simply stand idle for want of incentive to function, an incentive which should come, as I say, through the ear.

There are, it is true, dumb children who are not deaf. An annotation in the *Lancet* of 1906,[3] described such cases, and attributed the condition to a lack of normal brain spontaneity that can be co-ordinated in imitation of movements. Other speechless individuals who hear, may be simply manifesting what is clinically known as aphasia; while others may be speechless from disease or destruction of the speech organs. These obviously are not deaf mutes.

On the other hand, there are deaf people who can speak. But either they have become deaf after they learned to speak,—and it is a matter of experience that, if deafness does not come on, in a child of average intelligence, before the age of 7, he will usually retain the speech he has already acquired, and even add to it;—or, if the deafness developed earlier, then the speech is the result of special training.

So defined, then, deaf mutism is not a precise scientific entity, for some deaf mutes can hear something; and some deaf mutes can speak, or utter sounds. It is right, therefore, to speak of *total deaf mutism*, in which the victims are irresponsive to every auditory sensation, and *partial deaf mutism*, in which they can, not only make out noises, but also, it may be, words, if they are spoken loudly.

The latter, the partial deaf mutes, are the more numerous

by a long way. Kerr Love's observations led him, in 1896, to the view[4] that not more than 7 to 20 per cent. of deaf mutes are quite deaf to sounds conducted through the air, while only a still smaller proportion are quite deaf to sounds conducted by bone; and that hearing for speech, to a useful extent, exists in from 10 to 27 per cent.

MORBID ANATOMY AND CLINICAL FINDINGS.

On deaf mutism there is a considerable amount of evidence available, extending from the time of Herodotus,[5] and including records of examinations made with the speculum and tuning-fork, and of post-mortem dissections. Of the latter, the later records are naturally the more valuable, because until methods of precision came to be applied to the examination of delicate organs like the inner ear, the findings could be in no sense complete.

I shall hurriedly run over the more important points contained in these reports, and refer, at the same time, to those which any of us may observe in a clinical examination of a deaf mute. The abnormalities that are not often seen can quite well be passed over.

(*a*) *External Ear.*—The obstructions from various causes that may be present in the external ear are not in themselves of moment, but they may be of value in suggesting the possible presence of deeper malformations.

(*b*) *Middle Ear.*—The middle ear, however, is frequently profoundly altered, but by no means always. In regard to the membrana tympani, for example, a large number, even 30 to 50 per cent., are normal;[6] in many others the abnormalities are of comparative unimportance, being such as are usually found associated with the presence of enlarged tonsils and post-nasal growths. In a minority of cases, there is evidence of past or present suppurative disease, perforations, more or less complete absence, scars,[6] and calcareous deposits.

Further, the ordinary contents of the middle ear, the ossicles, muscles, ligaments, etc., often show destructive changes. All the ossicles may be absent, or two, or one. More frequently ankylosis of the ossicles has occurred, either bony or fibrous. The stapes is the bone most often affected, and has been found ankylosed in the fenestra ovalis in one-

eighth of all the dissections.[7] This is similar to the condition which Politzer has demonstrated in the disease formerly known as "dry middle ear catarrh," but now called "otosclerosis."

The inner wall of the middle ear, which is also the outer wall of the inner ear, is, however, the most noteworthy, and here are found the most frequent seats of abnormalities. It is in this wall, you remember, that the two windows, the fenestra ovalis and the fenestra rotunda, are situated, opening, in the dry skull, into the vestibule of the labyrinth and into the cochlea respectively, from which in health, however, they are separated, in the case of the fenestra ovalis by the foot-plate of the stapes and its ligament, and in the case of the fenestra rotunda by the secondary tympanic membrane. On this wall, too, is the promontory of the middle ear, caused by the projection of the cochlea; and behind and above the fenestræ are areas corresponding with the semi-circular canals. It is the round window, or the niche leading to it, that one most commonly finds affected; perhaps it is wanting, or filled up by osseous masses. Indeed, one-fourth of all the dissections, which have yielded positive results, exhibited anomalies in and around the fenestra rotunda. The oval window also may be similarly affected, though it is not closed so frequently, except in the cases of ankylosis of the stapes.

The middle ear cavity, including the mastoid cavity and the Eustachian tube, may be occupied by deposits of osseous and other masses; and the other boundaries of the middle ear and the walls of the mastoid may have undergone destructive changes.

Nearly all these middle ear conditions are explicable as the result of inflammatory processes, catarrhal sometimes, but generally suppurative, and then of a peculiarly destructive type.

(*c*) *Inner Ear.*—In investigating the changes in the inner ear, one can use only the method of post-mortem dissection, except in quite exceptional cases. Either the whole of the labyrinth, or only a part may be the seat of abnormality. If only a part is affected, it is most commonly the semi-circular canals, next comes the cochlea, and least often it is the vestibule. More than half the dissections, yielding positive

results, show changes in the semi-circular canals,[8] while, in a fifth of such dissections, they are the only part of the labyrinth affected.[8] This is somewhat surprising, when one remembers that the chief function of the semi-circular canals is equilibration, not audition.

The abnormalities present may be entire absence of the part concerned, either as the result of defective development, or, according to Mygind,[9] through replacement of the normal structure by bony tissue resulting from an otitis intima. Or the cavities may be filled with caseous or calcareous masses or fibrous tissue; or they may be reduced in size by hypertrophy of the walls, bony or periosteal; or increased in size by atrophy of the walls or destruction of the contents. In other cases, the osseous labyrinth may be normal, while the membranous labyrinth is the seat of the abnormalities—absence or alteration.

(*d*) *Auditory Nerve.*—Leading to the inner ear is the auditory nerve, and this also is frequently affected in deaf mutes. It may be completely absent. Michel[10] recorded a case in which there was no trace of it right up to the interior of the fourth ventricle; as the internal auditory meatus was so narrow as only to admit of the facial nerve, and the labyrinth was entirely lacking, it is probable that this was a congenital absence of the nerve, with consequent non-development of the labyrinth. In other cases, the nerve has been present but attenuated; while, occasionally, there have been abnormalities at the origin of the nerve, for example, complete absence of the striæ acusticæ.[11] Of all the nerve changes, however, the most frequent is complete or partial atrophy or degeneration of the trunk or its branches; indeed, one-eighth of all post-mortem examinations which have given positive results show this change.[11] It is to be noted, however, that, though in many cases of deaf mutism the auditory nerve has been functionless for so long, it is not always atrophied.

(*e*) *Central Nervous System.*—Finally, one finds that the central nervous system is but seldom affected in pure deaf mutism; and though changes have been found in the fourth ventricle, cerebellum and cerebrum, it is doubtful whether they could have any causal connection with the deafness.

PATHOLOGY.

Now, how does the pathologist explain all these changes ?

Obviously, the destructive changes in the middle ear are similar in kind to those we regularly see in chronic suppurative otitis media. Other of the changes are like the results of non-suppurative affections of the ear, and many of them appear to date from an early period.

As to the abnormalities in the inner ear, a great number of them are to be explained by the exceptional severity of the middle ear affection, leading to extension inwards. Those who were present at the meeting of the British Medical Association at Exeter last year (1907) would note that Dr. Milligan, during the course of a demonstration on Labyrinthine Suppuration, indicated that the vast majority of cases of labyrinthine suppuration are secondary to septic disease of the middle ear, though in only from 1 to 2 per cent. of cases of chronic septic otitis media does labyrinthine infection result.[12]

If the disease, suppurative or non-suppurative, is especially marked on the inner wall, it may lead to the deposit of fibrous tissue, and then of osseous tissue, in the niches of the fenestræ ; these, as a result, become functionless, and sound waves are no longer transmissible to the perceptive apparatus.

But if the inflammatory process is destructive, as it often is, inflammation and suppuration may spread through the fenestræ, or through the promontory, or through some fistulous track in the external semicircular canal, into the labyrinth itself. If the attack is acute enough, death may be the result ; if it is not so acute, inflammatory deposits may be made in the labyrinth, and these, becoming transformed into osseous tissue, obliterate, in part or in whole, the cavity or cavities concerned.

On the other hand, the labyrinthine condition may be due to spread from within the cranial cavity. Meningitis, for example, extending along the internal auditory meatus, may lead to changes in the labyrinthine spaces similar to those set up by inflammatory processes in the external parts. So, in this way, too, osseous deposits may occur in the labyrinth, and abolish the function of the auditory nerve endings.

ANAMNESIS.

The date, at which these various changes make their

appearance, cannot usually be ascertained. Mygind states emphatically that, in post-mortem examinations, it is often a matter of impossibility to decide between congenital changes and those acquired after birth.[13] The ear and brain are exposed to inflammatory attacks *in utero* as well as after birth, and similar thickenings and deposits may follow. So one is driven to the question of the history of the deaf mute, in order to obtain information as to whether a particular case has been congenital or acquired.

How long has the deafness existed? From birth? Or did it come on during infancy, or early childhood? Nearly always the mother's observations are the only observations available, in regard to these questions. And though any single report may be untrustworthy, in all probability the sum total of reports may be accepted as moderately correct. For, to quote Kerr Love,[14] though the difficulties of finding out how long the deafness has existed are often great, it yet happens that the mistakes affect the totals in opposite directions. An infant, whose congenital deafness has not been recognised, may have, within a few months of birth, a severe illness, and this illness may be supposed to have caused the deafness. On the other hand, an infant with good hearing may, almost from birth, have an insidiously coming-on deafness, which, when it is recognised, is supposed to have always been present. Taking one with the other, therefore, it is probably safe to accept the modern estimates, which ascribe about 50 per cent. of deafness to congenital causes, and about 50 per cent. to acquired. Inquiries made by Love and Addison, about 12 years ago, amongst a large number of English schools for the deaf, led them to the opinion that an average of 60 per cent. had been born deaf. Bezold, in his book on *Deaf Mutism*, published in 1902, records that out of the deaf mutes, who had passed through his hands in Munich, 100 were congenital, and 119 acquired.[15] Of 10 deaf mutes, of whom I have records during the present year, five were said to be congenital, and five acquired.

(*a*) CONGENITAL DEAF MUTISM.

Now, when one examines the history, and also the family history, of the congenitally deaf, one finds two facts, which

occur sufficiently often to be considered in the light of effective causes of deaf mutism, viz., heredity, and consanguinity of parents.

(1) *Heredity.*—The opinions of authorities upon heredity as a cause of deaf mutism vary widely. Kramer, of Berlin, whose work on *Aural Surgery*, in 1863, is amongst the publications of the New Sydenham Society, said,[16] categorically, "Deaf mutism is not a hereditary disease." It is a British aurist, the late Sir William Wilde, of Dublin, who lays most stress upon the hereditability of deaf mutism,[17] and this is the view that is coming to be widely held.

Perhaps the difference in the conceptions of heredity, held by the different observers, accounts for the divergent opinions. It is undoubted, for example, that extremely few deaf mutes are the children, immediate children that is, of deaf mute parents. The latest testimony available, Bezold's, is in accord with former authorities, to the effect that among all his cases of congenital deaf mutism, not one was an instance of direct hereditary transmission. My own experience of 11 cases of deaf mutism seen this year, concerning whom I inquired on this point, gave one case of deaf mutism with deaf mute parents.

But if one includes, under the idea of heredity, not only the direct ancestry, but also the collateral family tree, one finds that repeatedly the abnormality of deaf mutism appears. It is least frequent in the direct ascending line (grandparents, parents); more frequent in the collateral branches (great uncles, great aunts, uncles, aunts, grandparents' cousins, parents' cousins, cousins, and second cousins); and most frequent by far among the brothers and sisters of the deaf mute.[19] Mygind's Danish records, referring to over 500 deaf mutes,[20] show that about one-fifth of the whole had deaf and dumb brothers and sisters, and about one-fifteenth had deaf and dumb relations in more remote degrees. The last fact may be stated in another way, namely, that there was one deaf mute relative (parents, brothers, and sisters not included) to every sixteenth deaf mute. Certainly deaf mutism is not so common amongst the relatives of normal persons as that.

Various considerations may influence the heredity of deaf mutism, just as they influence the heredity of other abnor-

malities. "The special character of the parent is not uniformly transmitted to the offspring,"[21] it may be present in only a few of them, or an entire generation may be unaffected.[22] This is in accordance with the theory of reversion to the character of a more remote ancestor. Other considerations, which we know little or nothing of, may influence the appearance or suppression of deaf mutism. In the section of Pathology of the British Medical Association, meeting in 1905 at Leicester, Dr. C. J. Bond[22] presented a note in which he compared deaf mutism as a hereditary disease with hæmophilia, and illustrated, by genealogical trees, the point, that when deaf mutism crops up in a family, it tends to affect one sex only in any one family. In calculating, therefore, the chances of deaf mutism in a family, in which it is to be feared, our view must not only include the immediate progenitors, but the whole family antecedents on both father's and mother's side, "for the hearing members of a deaf mute connection send down the tendency to deafness with as great certainty as the deaf members."[23]

2. *Consanguinity of Parents.*—This, like heredity, is a much disputed matter, some authorities asserting that it has no deleterious influence, while others, prominent amongst whom is Wilde again, claim that it is paramount amongst the predisposing causes of deaf mutism. Those who contend for the harmlessness of consanguinity, point to the admirable results of inbreeding amongst domestic animals. The conditions, however, as Kerr Love[24] shows, are not similar in the two cases. For amongst sheep and cattle, the animals used are perfect specimens; in the case of man, the individuals have practically always some ill condition or tendency. It may be that it is these inherent weaknesses that lead to the bad results, and not the consanguinity *per se*, and this is the view stated by the Glasgow aurist.[25] In practice, the evil influences predominate in nearly all such unions casually arranged, and probably, he thinks, one must look upon the appearance of deaf mutism as only an example of degeneration, ranking therefore with other signs of degeneration in a family.

Sir Arthur Mitchell,[26] whose investigations were made in 1865, speaking of deaf mutism, after he had examined over 400 deaf mutes, stated that 1 in every 16 had parents who

were blood relations. If, said he, cousin marriages have no influence in the production of this result, then such unions in the general community ought to be to others in the proportion of 1 to 17. Such a proportion must be too great; the average for Great Britain is probably not more than 1 to 60 or 70.

Mygge,[27] working in Denmark, says that in that country consanguineous marriages may be supposed to represent about 3 to 4 per cent. of all marriages, yet 6·75 per cent. of deaf mutes, admitted into the Royal Deaf and Dumb Institution in Copenhagen, were the result of such marriages.

It is a striking fact that deaf mutism is more common amongst Jews than amongst Protestants or Roman Catholics, and that it is more common amongst Protestants than amongst Roman Catholics. This coincides with the customs that prevail amongst these different religionists in regard to marriages; for amongst the Jews intermarrying largely obtains; Roman Catholics, however, discourage the marriage of cousins, while Protestants, as is well known, permit such marriages.

Interesting examples of communities, which lend support to either view, as to the influence or non-influence of consanguinity of parents in producing deaf mutism, are cited by Kerr Love.[28] The island of St. Kilda, for example, where intermarrying has gone on for centuries, is said to have no history of any case of deaf mutism. Martha's Vineyard, on the other hand, an island in the southern part of Massachusetts, is an example of the opposite condition, for there, out of 500 inhabitants, in 1880, there were 20 deaf mutes.

A possible explanation of these anomalies, an explanation which I have never seen referred to before, may perhaps be found in a more exact investigation of the relationships of the parents. It was suggested to me through reading a paper[29] about 12 years ago by Mr. Basil H. Thomson, now Governor of H.M. Prison at Wormwood Scrubs, in which were described in detail the marriage customs of the Fijians. It appears that cousin marriages in Fiji are considered from two aspects :—

(1) Where the contracting parties are the children of two brothers, or of two sisters;

(2) Where the contracting parties are the children of a brother and a sister respectively;

and Mr. Thomson showed, in his paper read before the Anthropological Institute, that "from the Fijian point of view, the relationship between children of two brothers, or of two sisters, is exactly the same as the relationship between children of the same parents," and therefore marriage between two such children is strictly forbidden. On the other hand, cousins of opposite sex, of whom the father of one is brother to the mother of the other, are said to be "concubitants," that is to say, marriage between them is not only encouraged, it is obligatory. Mr. Thomson then detailed his examination of the census figures, taken with a view to the study of results of Fijian marriages, from which he found that, as to both fecundity and vitality of offspring, the marriages between concubitants are greatly superior to those between relations (not concubitants), or between fellow-townspeople (not related), or between natives of different towns. In contrast with this, it appeared that, as to fecundity and vitality of offspring again, marriages between relations (not concubitants) are greatly inferior to those of any other class.

This consideration, applied to the investigation of deaf mutism, means the preparation of entirely new statistics; and if this is done, it may reconcile the present discordant opinions.

If this position is accepted, namely, that about 50 per cent. of the cases of deaf mutism are the result of marriages, either—

(1) amongst those who have cases of deaf mutism in their families, either direct or collateral, or

(2) amongst those who are blood relations,

it is the duty of general practitioners, a duty which they will not seldom have an opportunity of practising, to do all in their power to discourage such marriages. This is, of course, a counsel of perfection, but there *are* people amongst the deaf, as amongst the hearing, who are prepared to sacrifice their pleasure to their duty in this respect, if only they know clearly what that duty is. Others, of course, will not hearken to us or to anyone else, and as at present the State is not ready to forbid such marriages, we are all more or less helpless. All that we can do therefore is, as Kerr Love concludes,[30] to see distinctly before us the goal to be attained to, and try to encourage others to aim at it also.

(*b*) ACQUIRED DEAF MUTISM.

So much for the congenital deaf mutes. What, finally, about the 50 per cent. of acquired cases of deaf mutism, that is to say, the cases of deaf mutism who began life hearing, and possibly attained a slight degree of speech, but who, as the result of some disease which occurred early in life, became deaf and dumb ?

(1) BRAIN diseases play an important part in the origin of deaf mutism. Hartmann[31] attributed 38·8 per cent. of the cases to brain disease; modern British statistics give 24 per cent.[32]

Of brain diseases, the most potent undoubtedly are *meningitis simplex* and *epidemic cerebro-spinal meningitis*.[33] They both act by spreading outwards from the original seat of the disease to the labyrinth; occasionally, the epidemic disease produces a preliminary inflammation of the middle ear. In epidemic cerebro-spinal meningitis, it appears that usually the invasion of the labyrinth occurs suddenly, and in the course of the first two weeks,[34] and it is probable that the disturbances of equilibrium, which are present, are due to the inflammatory processes in the semicircular canals.

(2) Next to brain diseases come acute infectious diseases, in which category, of course, epidemic cerebro-spinal meningitis may be included. Of the others,—

Scarlet Fever is far and away the most virulent, statistics varying from 7 to as high as 42 per cent. in accordance with the areas from which they have been obtained.[35] Scarlet fever causes deafness by inflammation of the middle ear, of a type having a marked tendency to destroy the mucous membrane and osseous walls and contents of the tympanum. This middle-ear inflammation arises most frequently by propagation through the Eustachian tube, but may appear independently. From the tympanum, the inflammation may spread through the fenestræ, or by way of the vessels, to the labyrinth, and cause a partial or total destruction of the membranous contents of that organ. It is said that the period, when the deafness shows itself in the majority of cases, is the period of desquamation.

Measles, to which are attributed 1·4 to 8 per cent.[36] of

deaf mute cases, acts in a similar manner to scarlet fever. Perhaps more often, however, the labyrinthine disease may develop independently of middle-ear inflammation.

Diphtheria, though not often mentioned as a cause of deaf mutism, may possibly be the forerunner of some of the cases, and its non-mention may be because it has been of a type so mild as not to be recognised.

Small-pox, *Chicken-pox*, *Erysipelas*, *Influenza*, *Whooping-cough*, *Mumps*, and *Typhoid Fever* have all been answerable for occasional cases of acquired deaf mutism.

(3) Finally, some CONSTITUTIONAL DISEASES may be the origin of deaf mutism. Of these syphilis is to be especially mentioned, for, though few cases are attributed to it in statistics, some observers, including Sir Wm. Dalby, think that it is a common cause of the condition.[37]

The practical outcome of the perusal of these causes of acquired deaf mutism is evident, and there is no need to emphasise it:—The acute infectious diseases come to us for treatment, and in many cases, not in all, we can do something, as we are doing, to prevent the extension of throat and nose inflammations to the middle ear; and, where the middle ear has become invaded already, something to check the destructive processes, which are so liable to follow, and also the involvement of the inner ear. But we all know well that, in spite of all treatment, in many cases the ear cannot be saved.

And so the end of this article is simply a "word to the wise." Give attention to the treatment of all diseases of the ear, and especially to those occurring in the course of the general diseases, which at times leave such disastrous results.

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